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Circadian Variations of Ventricular Arrhythmias and Sleep-Disordered Breathing in HF Patients

We read with interest the study by Patton et al. (1) on the unexpected absence of typical circadian variation of ventricular arrhythmias observed in the SCD-HeFT (Sudden Cardiac Death in Heart Failure Trial). Sleep-disordered breathing (SDB), broadly categorized into obstructive and central sleep apnea, has been associated with increased cardiovascular morbidity and mortality. Cardiac arrhythmias are responsible for some of the higher cardiovascular morbidity and mortality rates observed in patients with SDB. The association between atrial fibrillation and SDB is well established, although the association between SDB and life-threatening ventricular arrhythmias also seems plausible. Obstructive sleep apnea predicts sudden cardiac death independently of other well-established risk factors (2), and, unlike the general population, patients with SDB have a higher incidence of sudden cardiac death during sleep (3). Studies consistently report an SDB prevalence of ≥50% in the chronic heart failure population. The prevalence of SDB in patients with an implantable cardioverter-defibrillator (ICD) ranges between 57.8% and 66.3% (4,5). In a cohort of 472 ICD patients with heart failure receiving cardiac resynchronization therapy, a significant risk enhancement of ventricular arrhythmias and appropriate ICD therapies owing to both central and obstructive sleep apnea was found (5). Importantly, for heart failure patients with a primary inappropriate ICD therapies (4). Patton et al. (1) observed an increase in the onset of ventricular arrhythmias during sleep in patients with an ICD and SDB. Data on SDB for patients enrolled in the SCD-HeFT were not reported. Thus, it is our opinion that the observed deviation in circadian variation of ventricular arrhythmias reported by Patton et al. (1) may be influenced, at least in part by the presence of SDB, a very prevalent condition among heart failure patients with an ICD.

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REPLY: Circadian Variations of Ventricular Arrhythmias and Sleep-Disordered Breathing in HF Patients

We appreciate the interest of Dr. Arias and colleagues in our study of circadian and septadian patterns of implantable cardioverter-defibrillator therapy in the SCD-HeFT (Sudden Cardiac Death in Heart Failure Trial) population (1). In their Letter, they relevantly highlight the importance of sleep-disordered breathing as a trigger of ventricular arrhythmias and implantable cardioverter-defibrillator therapies. Both central and obstructive sleep apnea exert strong effects on the autonomic nervous system and are known to be proarrhythmic (2).

We agree with Dr. Arias and colleagues that sleep-disordered breathing is an important and increasingly recognized trigger of arrhythmias (3). Unfortunately, we do not have information on the presence

or absence of sleep apnea in SCD-HeFT and were therefore unable to disentangle this likely complex relationship. However, we note that the overall cohort and several subgroups analyzed still showed a typical nadir in arrhythmia therapy from 12 AM to 6 AM, which is the conventional sleep time period with increased sudden death risk in the sleep-disordered breathing population (4). Interestingly, this nadir was not significant in the nonischemic heart failure group, the class III heart failure group, in subjects younger than 50 years, or in the beta-blocker group (1).

Further clinical studies to elucidate the relationship between sleep-disordered breathing and ventricular (and atrial) arrhythmias to design preventive therapies will clearly be of great interest (5).

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Please note: Dr. Patton has been a site investigator in a Cameron Health clinical trial. Dr. Poole has received lecture fees from Medtronic, Boston Scientific/Guidant, Biotronik, and St. Jude Medical; consulting fees from Physio Control; holds equity in Cameron Health; and has served on the Advisory Board of Boston Scientific. Ms. Anderson has received consulting fees from Boston Scientific, Inc. Dr. Mark has received consulting fees and research grants from Medtronic. Dr. Lee has received research grants and consulting fees from Medtronic. Dr. Bardy has received research grants from the National Heart, Lung, and Blood Institute and St. Jude Medical; and holds equity in and intellectual property rights with Cameron Health. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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